study reports a 34 gram birth weight loss for ETS exposure at home and a 20 gram increase of birth weight for ETS exposure in the workplace (Ahlborg and Bodin, 1991).

A number of problems with these studies have been recognized. Using paternal smoking as an index of ETS exposure could lead to substantial misclassification, especially if mothers of underweight infants may be more inclined to put the blame on someone else. (Lazzaroni et al., 1990). It is also well known that an average close to 5% of self reported nonsmokers are in fact or have recently been smokers, a misclassification tendency that may be even more prevalent in mothers experiencing the guilt of smoking during pregnancy.

The presence of so many biases and confounders is bound to produce a confused picture, For instance, a study of over 13,000 women observed that neonatal mortality in the offspring of nonsmokers married to smokers was higher than for the offspring of mothers actively smoking during pregnancy (Yerushalmy, 1971). Yet, studies cited in this section and other studies did not find any association of paternal smoking with prematurity or perinatal mortality (Tokuhata, 1968).

Intrauterine Growth Retardation (IGR). Although it is established that smoking reduces birth weight by an average of 200 grams, the association seems to disappear at smoking rates of less than 13 low yield cigarettes per day (Peacock et al., 1991). In any event, a review by a Harvard Medical School group states: " The medical significance of low birth weight is not totally understood...Morbidity among infants of smokers due to low birth weight alone is not clearly documented." (Werler et al., 1985).
Identified risk factors for low birth weight are: job stress, preconceptional and prenatal care, iron deficiency, nutritional deficits, maternal hypertension, alcohol consumption, illicit drug use, HIV infection, dieting, injections and medications, and oral contraceptives (Bendich, 1993; Schieve et al., 1994).

Among mechanistic hypotheses, fetal hypoxia has been suggested (Meyer, 1978). A more convincing deduction comes from the well known observation that smokers eat and weigh less. In this light, it has been determined that birth weight deficit may not be directly related to smoking, but may result from reduced nutritional intake of pregnant women who smoke. This conclusion is supported by the evidence that proper nutritional management and supplementation appears to prevent birth weight loss and a number of its sequelae. (Rush et al., 1980; Haste et al., 1991; Keen et al., 1993; Brown,

1993; Jameson, 1993). Significantly, intervention trials were successful in controlling smoking prevalence in pregnant subjects, but this reduction generally failed to result in increased birth weights, in the absence of proper nutritional management (Gillies, 1992).

A more recent study of 50,000 births in Cardiff, Wales, also indicates that birth weight loss became significant only for subjects of low socioeconomic class, who were comparatively less well nourished (Rush et al., 1990). The major significance of maternal nutrition in regard to birth weight and other pregnancy outcomes is now fully recognized (Keen et al., 1993; Brown, 1993; Jameson, 1993). Therefore, the implication is that ETS could not pose a low birth weight risk, since ETS is not known to adversely affect the nutritional status of exposed nonsmokers.

Spontaneous abortion/miscarriage. Some studies have reported a slightly increased risk of spontaneous abortion in active smokers (Himmelberger et al., 1978; Kline et al., 1977). However,in a large study of 32,000 women the risk of spontaneous abortion became insignificant after adjusting for alcohol consumption (Harlap and Shiono, 1980). Reviewers listed a number of confounding variables that prevent reliable conclusions (Stillman et al., 1986; Harlap, 1986).

Perinatal mortality. A recent review adversary to smoking states that "...the effect [of active smoking] on perinatal mortality is likely to be very small, especially for those babies weighing >3000 grams." (Stillman et al., 1986). In fact, epidemiologic results are very much conflicting, with some studies reporting and others not reporting an association (Peterson et al., 1965; Yerushalmy, 1964).

Smoking actually has been reported to reduce toxemia in the newborn (Rantakallio, 1978). A study of 605 women also found that maternal smoking was associated with the absence of respiratory distress syndrome in newborns (Curet et al., 1983). The latter observation is reinforced by evidence that maternal smoking seems to accelerate the fetal maturation of the lung (Liberman et al., 1992).

Malformations. Most studies have failed to implicate maternal smoking as a cause of malformations (Cordier et al., 1992; Tikkanen and Heinonen, 1991; Hemmiki et al., 1983; Evans et al., 1979; Sachs, 1989). This conclusion has been reached by most reviews adverse to smoking. For instance, the just mentioned review by a Harvard Medical School group provides a long list of the possible confounders and biases in such studies,

and states: " [The] contradictory results make it difficult to conclude that smoking is an important teratogen, or that it is entirely free of teratogenic effects." (Werler et al., 1985).

A review from the American College of Obstetricians and Gynecologists writes: "Most studies have not found a relationship between smoking in pregnancy and birth defects..." (ACOG, 1993). Moreover, recent symposia highlighted the role of nutritional deficiencies in outcomes of malformation, a powerful confounding factor if smoking expectant mothers should eat less (Keen et al., 1993; Wald, 1993).

Studies also found that abortions of *nonsmokers* were 40% more likely to show chromosomal abnormalities than abortions of smokers, thus indicating that smoking may not have genetic effects. (Kline et al., 1983). Other studies have shown that maternal smoking is associated with a significantly *decreased* risk of Down syndrome development (mongoloid retardation) (Kline et al., 1983; Cuckle et al., 1990).

In very large and recent studies no risk of malformation was associated with paternal smoking (Seidman et al., 1990; Savitz et al., 1991). This conclusion is reinforced by studies of subjects participating in programs of in vitro fertilization, where many of the confounders would be well controlled (Pattinson et al., 1991).

Sudden Infant Death Syndrome (SIDS). Many confounders could be responsible for the reported association between SIDS and maternal smoking, among them the recently emphasized paractice of laying infants to sleep in a prone position on bed surfaces that are too soft. An editorial in Lancet stated that there is no evidence that reducing smoking in mothers would affect the incidence of SIDS or infant mortality. (Lancet, 1985). Part of the problem is that "...it is now believed that SIDS is not a single entity, and that a number of different causal mechanisms are involved." (Nicholl and O'Cathain, 1992). Although some studies report an increased SIDS risk, others report reduced risk. (Matthews and O'Brien, 1985).

Risk of cancer. It should be argued again that studies of maternal smoking before and during pregnancy -- the exposure index for most childhood studies reviewed -- relate to active smoking and are improperly included in a review of possible ETS effects.

Neutel and Buck report on all childhood cancers as well as specific sites. On overall cancers, their Table 1 reports that for overall cancers the risk may be increased for

smokers of less than 1 pack/day, but *decreased* for smokers of more than 1 pack/day, and this both for the Ontario and British cohorts. Table 3 suggests that increased risk is principally confined to the first two years of age -- more unlikely to be ETS-related -- and reverses to considerable protection by age 5-7 (Neutel and Buck, 1971).

The Golding et al. study reports a RR=1.58 for smokers of over 5 cigarettes/day, but a RR=0.64 for smokers of less than 5 cigarettes/day, a possible indication of threshold for *active smoking* exposure since the study deals with smoking mothers (Golding et al, 1990). The Pershagen et al. study of over 500,000 Swedish children is probably the best available and by any possible restructuring of the data produces relative risk estimates below 1 (Pershagen et al, 1992).

Of the more questionable case-control studies, the Manning and the Stewart et al. studies produced no evidence of elevated risk (Manning, 1957; Stewart et al., 1958). The Stjernfeldt et al. studies dealt with mothers smoking during pregnancies, used diabetic children as controls, were poorly controlled and are of questionable meaning (Stjernfeldt et al., 1986a,b). Subsequent large studies by McKinney et al. and by Buckley et al. did not show risk elevation(McKinney et al., 1987; Buckley et al., 1986). John et al., on the other hand, reported a small overall risk elevation that could have been opposed by adequate adjustment for social class (education) of smoking fathers (John et al., 1991). Indeed, studies that attempted to separate ETS exposures and exposures from active smoking during pregnancy provide no evidence of ETS effects.

Virtually all studies of brain cancer risk refer to exposures by mother's active smoking during pregnancy, and therefore are not relevant to ETS exposure and inferences, and do not support an effect of mothers smoking during or prior to pregnancy. Gold et al. suggested and then refuted with a better study the notion of an association for brain cancer comparing mothers who stopped smoking or not during pregnancy (Gold et al., 1979, 1993). This suggestion has been further negated by two recent studies (McCredie et al., 1994a,b). The latter studies suggest that mother smoking may strongly *protect* against brain cancer risk, although it provides an additional – albeit baffling – suggestion of an association with father's smoking.

From these data, it appears that maternal smoking does not increase and may even reduce the risk of brain cancers. There is equivocal suggestion that the risk may increase if the father smokes. A biological plausibility of an ETS involvement in this second

hypothesis is obviously not supported in view of no effect or potentially protective effect for mother's smoking. Causal factors other than ETS would have to be invoked and investigated, if the association is indeed real and not spurious.

On the issue of ETS and leukemia risk, it should be noted that an association of active smoking and leukemias is by no means clear. Some earlier studies have reported an association, but more recent, better controlled studies show no association or are quite equivocal. The British Doctors study -- widely reputed as one of the largest and most accurate -- reported a positive dose response for *protection* of active smoking against marrow and reticuloendotelial cancers (Doll and Peto, 1976). The Third National Cancer Survey by the National Cancer Institute found no connection between active smoking and risk of leukemias (Williams and Horm, 1977). The Kabat et al. study -- with one of the largest number of leukemia cases -- reported no risk elevation against cancer controls, and a *protective* association when compared against subjects without cancer. The study had the largest number of acute non-lymphocytic leukemias (ANLL), for which a negative risk was reported (Kabat et al., 1988).

The Cancer Prevention Studies (CPS I and CPS II) of the American Cancer Society indicate a consistent *reduction* of risk for lymphatic leukemias in current smokers. An observed reduced risk for myeloid leukemias in females of the CPS I survey contrasted with an elevation of risk in the CPS II survey. Risk for all leukemias was elevated except for females in the CPS I survey, where a substantial risk reduction was noted. No dose/response trend for myeloid leukemias was reported. The CPS II survey reported a reduced risk for myeloid leukemias for the highest cigarette consumption group of current smokers. (Garfinkel and Boffetta, 1990).

Brownson et al. report contrasting findings. For males, their study notes a modest risk elevation for smokers of less than 20 cigarettes/day, but a risk *reduction* for smokers of more than 20 cigarettes/day. The same inverse trend is apparent in females. The study suffers from a major problem, having used cancer patients as controls (Brownson et al., 1991). Based on the previous experience of Kabat et al. -- as noted above -- a control group of subjects without cancer is likely to have reduced the noted risks significantly or completely. Brownson et al. also conducted a more recent review and meta-analysis of most available studies -- excluding the Third National Cancer Survey report, for instance -- and reached the puzzling conclusion that "[t]he consistency, temporality, and biological plausibility of this relationship...support a causal relationship" of cigarettes

smoking and leukemia (Brownson et al., 1993). Indeed, such a conclusion appears possible only through a remarkably selective choice of data and of coincident reports.

As for ETS, leukemias, and children, the earlier studies of Manning and of Stewart et al. report no risk elevation (Manning, 1957; Stewart et al., 1958). The massive Pershagen et al. study is consistent with a reduction of risk (Pershagen et al., 1992). The Stjernfeldt studies suffer from the choice of diabetic children as controls (Stjernfeldt et al., 1986a,b, 1992). The same applies to the John et al. study that failed to appropriately control for the socioeconomic status (education) of smoking fathers (John et al., 1991). The remaining studies -- also with noted shortcomings -- are consistent with no elevation and possibly a reduction of risk (McKinney et al., 1987; Buckley et al., 1986; Magnani et al., 1990).

Respiratory problems in childhood. A number of studies have reported an association between maternal smoking during and after pregnancy and respiratory symptoms in their offspring. Most associations seem valid for respiratory symptoms in infants up to one year of age, but not thereafter. The reasons are unclear, since the associations could be explained by many concomitant factors besides ETS, such as socioeconomic status, diet, housing conditions, quality of parental care, maternal diet and health, and others. That the association generally does not hold for older children adds to the uncertainty of its meaning. ETS does not seem to be a factor in childhood asthma. Inconsistent and puzzling results are not uncommon: for instance, the National Child Development Study followed 8641 children from birth to age 16 and found strong associations between heavy maternal smoking during pregnancy and asthma, wheezing and bronchitis in their children. However, after controlling for several factors the study reported a reduction of risk for children of moderately smoking mothers. (Fogelman, 1980). Children of smoking mothers who were breast-fed had less respiratory symptoms than bottle-fed children of nonsmoking mothers (Pollock, 1992).

Samet, a well known critic of smoking,states: "evidence for association of involuntary smoking with childhood asthma is conflicting." Surprisingly, in the same paper Samet goes on to say: "Passive smoking has been shown to have adverse effects on the status of asthmatic children that are clinically significant". In reality, most of the confounding factors listed above also pertain to asthmatic symptoms. Reports associating ETS exposure with increased risk for middle ear and other ailments also suggest a significant interference from infectious agents that may be independently associated with ETS

exposure. Quoting again Samet: "presumably these illnesses are infectious in etiology and do not represent a direct response...to toxic components of ETS." (Samet, 1992). Puzzling questions are also raised by the apparent strong protection against adult lung cancer following exposure to ETS during childhood, as reported in some large recent studies (Brownson et al., 1992; Stockwell et al., 1992; Fontham et al., 1991).

Infant and child development. In a recent review generally unfavorable to smoking, Rush -- one of the most experienced authors in the reproductive effects of smoking -- begins with a warning about inevitable confounders and states that smoking mothers "are, on average, very likely to be...at a disadvantage, even after stratifying on any social class index...". He also states that "[w]hile it is necessary to control for such differences in analysis it is almost surely illusory to assume that it can be done fully." He goes on to say that "[i]n spite of such likely confounding, differences in cognitive and neurological development in childhood reported in many studies to be associated with parental smoking were only minimally controlled for social factors, and while they may have been highly significant statistically, they cannot be straightforwardly interpreted as having been caused by smoking." (Rush, 1992). The same reviewer concludes that "there do not appear to be dramatic, or even consistent behavioral effects on the neonate from smoking of their mothers other than less good auditory orientation. Whether such possible effects are associated with any long-term or permanent deficit is unknown." Also, that "there was no consistent nor strong pattern of deficit in infant or preschool child development among offspring of smoking mothers." In regard to cognitive development and achievement in school age children, Rush again states that although "there is a consistent pattern of depressed cognitive development and school achievement associated with maternal smoking during pregnancy...it appears beyond current knowledge to conclude that these associations were causally related to maternal smoking." Rush concluded that "[w]ether maternal smoking during pregnancy causes later behavioral abnormalities in the child remains an important and intriguing hypothesis, but from the available data it is not possible to judge whether a causal relationship exists."(Rush, 1992).

ODOR, IRRITATION, AND ANNOYANCE

Because of the weakness of epidemiologic inferences of ETS risk, opponents have successfully attempted to portray ETS as a major sensory and esthetic aggravation. Here, however, the intent to establish a cultural aversion is more evident than its circumstantial justifications.

There is no question that ETS can irritate, but only above certain threshold limits of concentration (Cain et al., 1987). The odor of ETS alone can be annoying. Human noses, however, are formidable chemical detectors down to concentrations in the parts per trillion range, and reactions to odors can have large psychosomatic components. Recent studies show that reaction to ETS odor changes from neutral to aversive depending on whether subjects are or not visually aware of the presence of active smokers (Moschandreas and Relwani, 1992). A prominent expert writes:

"People assess the quality of the air indoors primarily on the basis of its odor and on their perceptions of associated health risks. Whereas fear of adverse health effects of body effluvia once dominated such perceptions, fear of environmental tobacco smoke now dominates.
"...The relative health threats...may be quite the opposite of the resident's impressions. In the realm of odors and ventilation, however, any perception of a threat counts heavily. The layman may not know that bad smelling things are not necessarily dangerous...and that neutral or even good smelling things may in fact be dangerous. "Many people now perceive smoky air as a threat to health. The motivation to control it will therefore derive largely from this perception, much as the motivation to control body odor once derived from fear of its health impact." (Cain et al., 1987).

Besides, ETS can only be a relatively minor contributor to environmental irritants and odors, especially in workplaces. The major sources of such nuisances derive from new construction technologies, materials, equipment, and tools, as shown by the detailed description of indoor air quality determinant offered by the Proposed Rule itself.

THE SICK BUILDING SYNDROME

Modern air conditioning technology and energy conservation economies have resulted in nearly hermetically sealed work buildings and residential environments, often contributing to the "sick building syndrome".

The Proposed Rule and recent reviews and studies list many contributors to this syndrome: temperature and humidity, static electricity, lighting fixtures and intensities, noise and reverberation characteristics, airborne dusts and fibers, volatile organic compounds, and microorganisms (USOSHA, 1994; Skov et al., 1990; Brooks et al., 1991). By restricting attention to gases and particles, possible sources in typical buildings have been identified as adhesives, caulking compounds, carpeting, ceiling tiles, chipboards, particle boards, floor and wall coverings, paints, stains, varnishes, waxes, cleaning fluids, pesticides, appliances, carbonless copy paper, computers, video display terminals, duplicating machines, electrophotographic printers, photocopiers, microfiche developers, blueprint machines, printed paper forms, typewriter correction fluids, motors, hydraulic power equipment, heating and cooling equipment, lubricants, cooking, clothing, etc. (Brooks et al., 1991; Weschler et al., 1990). In general these studies refer to white-collar work conditions, and additional sources would have to be considered for specific and more congested occupational settings.

Recent studies also show that often indoor and outdoor concentrations of prevalent volatile organic compounds can be quite similar, suggesting significant external contributions to indoor air quality (Cohen et al., 1989). Inescapably, this evidence implies that the contribution of ETS must be small in the context of all sources of interference. Such a conclusion is in line with a report from the National Institute for Occupational Safety and Health, which traced only 2% of investigated indoor air quality complaints to problems where ETS might have been a possible component (Melius et al., 1984).

It is apparent that indoor air quality problems and the sick building syndrome stem both from objectively identifiable material sources, and from subjective anxieties and fears not rationally justified and nearly always the result of alarming reports uncritically broadcast by the mass media, as lamented by a report of the National Institute of Occupational Safety and Health (Melius et al., 1984).

CONCLUSION

Dose differentials and evidence of no-adverse-effect thresholds for active smoking preclude an inference of ETS risks, unless we are prepared to forgo all we have learned since Paracelsus about pharmacodynamic and kinetic discontinuities at low doses. Plausible ETS doses are thousands of times less than doses that appear to have no adverse effect in active smokers. Experimental reports in man or animals do not contradict this conclusion, which is reinforced by the equivocations of epidemiologic studies. The latter are impotent in controlling for a multitude of confounders, are plagued by irresolvable biases, and are consistent with slightly increased or decreased risk. Their only tenable summation is that ETS risks -- if at all real – are imponderable and beyond detection.

A case against ETS as a health risk cannot be made on scientifically objective grounds. Thus, an outright smoking ban in workplaces is unwarranted and would amount to unfair and alarmistic regulation, which could be only interpreted as a self-serving and arbitrary ruling. It could also divert attention from other numerous sources of indoor air quality deterioration, sources that by all accounts are far more significant than ETS.

Nevertheless, irritation and annoyance caused by ETS in nonsmokers need to be addressed as a matter of civic courtesy, despite their recently acquired psychosomatic and cultural overtones. The provision of smoking and nonsmoking areas in workplaces and adequate ventilation standards would be fully sufficient remedies, and a laudable reflection of equanimous and responsible policies.

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